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CONTENTS

The Growth of the Mandible . . . . HARRY SICHER 87

Bone Changes in the Jaw Caused by Renal Hyperparathyroidism . . JOSEPH P. WEINMANN . 94

Nutrition and Dental Health . . . . DOROTHEA F. RADUSCH . 105

Studies in Dental Nomenclature: A Series . . . . . HARRY LYONS 110

Editorials 113

Notes on the 1945 A. A. of P. Postgraduate Course . . . . . ROGER E. HUNTINGTON . 115

Publication in the JOURNAL OF PERIODONTOLOGY *is not to be understood as endorsement by the Journal or the* AMERICAN ACADEMY OF PERIODONTOLOGY *of opinions expressed in any article, abstract or extract. The object of publishing the various views is to present them without prejudice to the reader for his own evaluation.*

THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely will make the Journal a fund of extensive information, readily available to students, teachers, researchers, and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and clinical interest which are presented at the annual meetings of that society. Abstracts of important articles related to periodontology that appeared during the year of 1944 will be continued in the October issue.

We are grateful to all who have made available the subject matter from which the abstracts and copies of abstracts have been made, including the authors of the original articles, owners of the journals mentioned in the text of the abstract section, and those who have aided by contributing the material for this issue.

DENTAL LIBRARY

[Acute, necrotizing, ulcero-membranous gingivitis(?)]

Croupy Inflammation in the Margin of the Gums, and Its Effects. *By* Dr. Steinberger, of *Vienna.* Translated by George Henry, M.C.D.E., *London Dental Review,* October, 1861. *Condensed* in the *Dental Cosmos,* 3(6):320-323. Jan., 1862. Extracts from this follow.

"Croupy inflammation of the gums is simply referred to by most authors . . . as 'diphtheritis membranacea' . . . When the disease occurs in adults, the dentist often meets with it in its earlier stage, the patient being induced to consult him, partly owing to pain, partly to the fetid odor arising from the affection . . .

"The gum is inflamed, somewhat swollen, and very painful, and prone to bleed on being touched. In its early stage, the margin of the gums is found to be covered with a pale-gray, structureless membranous exudation, scarcely half a line in thickness, which sloughs off in flakes. The mucous membrane beneath is deprived of its epithelium, very vascular, and bleeds readily . . .

"The exudation takes place rapidly . . . It is accompanied with a very painful sensation . . . Feverish appearances are slight . . .

"As soon as the exudation is formed, it sloughs away in an ill-smelling ichorus mass, which corrodes the mucous membrane beneath, producing an ulcerated surface.

"The duration of this disease is dependent on its extent, the stage in which treatment is first resorted to, and, further, on the general state of the patient's health . . . Slight cases are curable in from eight to fourteen days, the more severe requiring often several months.

"Treatment.— . . . The first thing suggesting itself as favorable to a cure is extremely careful cleansing. The best plan is to remove the exudation before it is thrown off. This is readily effected, and that without giving much pain, by removing the exudation, especially from between the teeth . . . This done, the ulcerated surface is cleansed from blood by syringing with warm water; a little alum may be added when the hemorrhage is excessive . . . The wounded surface should be touched with caustic . . . apply it well between the teeth . . . The patient should frequently take warm mucilaginous decoctions in the mouth, which should be a cleansing and soothing kind . . .

"Besides this local treatment, the 'general' is confined to change of air, and, in the convalescent, a liberal diet . . . "

THE JOURNAL OF PERIODONTOLOGY

JULY, NINETEEN HUNDRED FORTY-FIVE

The Growth of the Mandible

By HARRY SICHER, M.D., *Chicago, Illinois**

THE GROWTH of the mandible has been the subject of several investigations in recent years. Brodie (1) studied the changes of the growing mandible by using serial cephalography. He emphasized the importance of the growth center in the mandibular condyle, which was first described by Toldt in 1884 (2). Brodie showed, furthermore, that the contour of the mandibular angle does not change appreciably during the growth of the child. Diamond (3), Sicher and Weinmann (4), and Weinmann and Schour (5) brought new evidence for a correlation between condylar growth and eruption of teeth; the increase in height of the mandibular ramus opens the space between upper and lower jaws into which the teeth erupt and the alveolar process grows.

In spite of these recent studies the significance of the peculiar growth mechanism of the mandible under normal and pathologic conditions has not yet been fully understood. It is the purpose of this paper to summarize our present-day knowledge and to draw conclusions from it which hitherto have been neglected.

If we start the description of the mandibular growth at an age of one to two years the areas of growth are already fairly restricted. The growth center in the condyle is represented by a cartilagenous disc covering the bony head of the mandible (Fig. 1); this cap of hyaline cartilage, however, cannot be compared to an articular cartilage because it is, itself, covered by a fairly thick layer of dense fibrous tissue. This fibrous tissue borders immediately onto the articular cavity and is in contact with the articular disc. Proliferation of the hyaline cartilage, and its replacement by bone, contribute both to the increase of the mandibular ramus in height, and to the increase of the over-all length of the mandible. The latter can be measured either by taking the distance between condyle and chin point, gnathion, or by measuring the distance from the gnathion to a point where a perpendicular line from the condyle meets the gnathion-gonion line (Fig. 2). The double effect of condylar growth is due to the fact that the condyle is obliquely implanted upon the body of the mandible by the obliquely ascending ramus.

By the condylar growth the over-all length of the mandible increases, not, however, the length of the mandibular body (gnathion-gonion). The con-

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Presented at the Postgraduate Course of the American Academy of Periodontology, W. K. Kellogg Foundation, Ann Arbor, Michigan, May, 1945.

dylar growth does not contribute either to the necessary increase of the antero-posterior width of the ramus itself. Here, appositional growth along the entire posterior border of the ramus is the means to adjust the width of the ramus and the length of the body to the growing height of the ramus (Fig. 3). Appositional growth at the tip and the upper borders of the coronoid process keeps pace with the heightening of the ramus, and resorption along the anterior border of the coronoid process and ramus corrects, at the same time, the antero-posterior dimension of the ramus and lengthens the alveolar space distally.

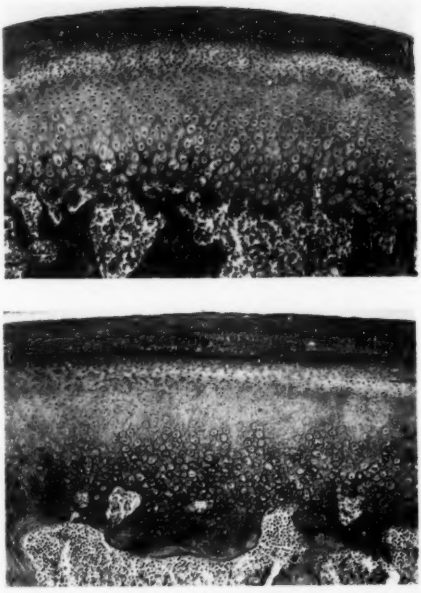

Fig 1. Sections through mandibular condyles showing typical endochondral ossification.
A. New born.
B. Young adult.
a Fibrous covering of mandibular head.
b Zone of appositional growth of cartilage.
c Hyaline cartilage.
d Bone.
(From Orban, Oral Histology and Embryology)

The growth of the mandibular body, measured from the lower border of the mandible to the free border of the alveolar process, increases almost exclusively by apposition of bone at the free borders of the alveolar process, growing into the space which is opened by the growth of the mandibular ramus in height. Apposition of bone at the lower mandibular border is negligible, apposition in the region of the chin serves not so much the mandibular growth as the modeling of the lower jaw. There is no doubt that corrective apposition at the outer surface of the growing alveolar process and modeling resorption from within, help to adjust the alveolar bone to the erupting teeth, especially during shedding of the deciduous and eruption of the permanent dentition. These growth phenomena, however, do not play a decisive role in achieving the adult dimensions of the mandible.

The important observation of Brodie that "the ascending ramus always meets the lower border of the body at the same angle" has often been misunderstood. Brodie himself points out that it is the definition of the "mandibular angle" which led to confusion. In the accustomed way, this angle was measured by placing the mandible on a horizontal table with a hinged leaf which is put in such a position that it touches the posterior border of the mandibular ramus at two points, near the condyle and near the angle of the mandible itself (Fig. 4). If we understand as mandibular or gonial angle, the angle at which the ascending ramus meets the lower border of the body, then the comparison of roentgenograms of the same child, at different ages, shows that this angle does not change. The "mandibular angle" of the textbook, how-

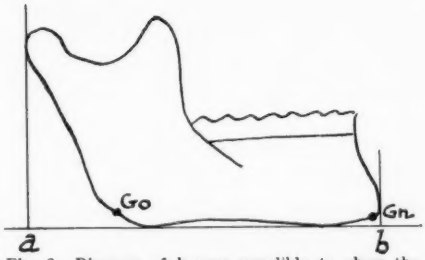

Fig. 2. Diagram of human mandible to show the difference between over-all length and body length.
ab Overall length.
go Gonion.
gn Gnathion.
go-gn Length of mandibular body.

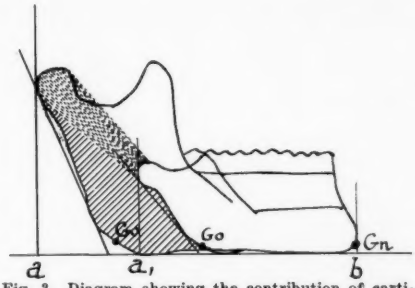

Fig. 3. Diagram showing the contribution of cartilagenous condylar and appositional bone growth in the development of mandible. Condylar growth (stippled) increases height of ramus and over-all length of mandible: ab and a'b. Appositional growth at posterior border of ramus (hatched) widens the ramus and increases length of mandibular body: go-gn. Mandibular angle decreases during growth (see Figs. 4 and 5).

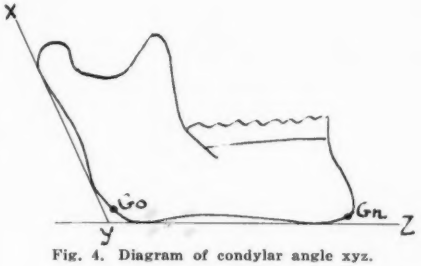

Fig. 4. Diagram of condylar angle xyz.

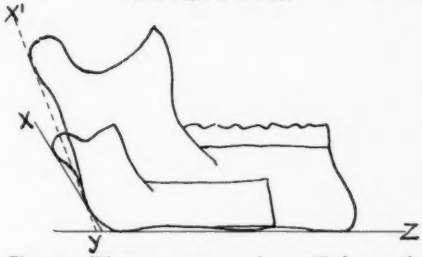

Fig. 5. Whereas contour of mandibular angle (gonial angle) remains unchanged, condylar angle xyz decreases during growth to x'yz. (In part after Brodie).

ever, does change (Fig. 5), a fact which Brodie expresses by saying that the condyle "is given off at a constantly higher level throughout the growth period although following a constant pattern."

It must be pointed out that the change of the mandibular angle has a very definite significance. This angle which might be better termed *condylar angle*, as against the *gonial angle* of Brodie, decreases during the growth of the mandible. It is an indication of the relation of condyloid process to mandibular body and dentition; it is, therefore, an indication of the relation of dentition to mandibular joint. Its change indicates the change in the mechanics of the mandibular joint and the muscles of mastication. The decrease of the condylar angle, furthermore, indicates a change of the direction of the plane of condylar growth to the body of the mandible. The older the child the less inclined is the condylar growth plane to that of the lower border of the mandible or to the occlusal plane. Of the two components of condylar growth —increase of the ramus in height and increase of the over-all length of the mandible—the latter is forced into the background by the change of the condylar angle.

In spite of the change in the relation of the two components of condylar growth, it is of great importance to realize that growth in height of the man-

dible and growth of the mandible in length are inseparable. A mandible which is too long will also have a ramus that is too high and vice versa. It has been pointed out that apposition of bone at the posterior border of the mandibular ramus contributes to the lengthening of the mandibular body. The two components, acting together to bring the mandibular body to its full length, i.e. condylar growth and growth at the posterior border of the ramus, are of an entirely different nature. In the condylar growth the growth of cartilage plays the leading role. It is the proliferation of the cartilage, and not its replacement by bone, which makes the mandible grow in height and over-all length, just as a long bone grows in length by proliferation of the epiphyseal cartilage. Replacement of the proliferated cartilage by bone, though indispensable for the proper function of the growing bone, does not contribute to the enlargement of the bone as a whole. In contrast to this, the growth of the mandible along the posterior border of the ramus is growth of bone by direct apposition of the new upon the old bone. This growth can be compared to the periosteal apposition of bone in the shaft of a long bone, which contributes to the transverse growth of the long bone or to its growth in thickness. The contrast between growth of cartilage and growth of bone, in establishing the relation between length and thickness of a long bone, is best expressed in the two types of the human body which we call the linear and lateral types. In the first, the entire body and, of course, the skeleton and especially the long bones of the extremities, are long and slender. In the latter type, body and bones are short and thick. In the first type, cartilagenous growth exceeds that of bone, in the latter, growth of bone exceeds that of cartilage. Applied to the mandibular growth, we see here the perfect analogy. If cartilagenous growth outbalances the growth of bone, the mandible will be long, the ramus high and narrow, the angle obtuse, in other words, the mandible of this type will show all the characteristics of that of a leptoprosope skull. If, on the other hand, growth of bone outdistances cartilagenous growth, the mandible will show the characteristics which we find in the euryprosope skull; the mandible will be short, the ramus low and wide, the angle will approximate a right angle.

These differences are magnified in cases of Class II and Class III, malocclusions. The mandible of a true Class II is too short. This mandible is also always characterized by too low a ramus and too small an angle. In these cases, the cartilagenous growth at the condyle is, for no known reason, impaired. Thus, the mandible will not reach its normal height. The bony growth, at the posterior border of the mandible, proceeds more nearly at a normal rate and, consequently, the ramus is wide and the mandibular angle nearly a right angle. That a mandible, which is too short, has, necessarily, too low a ramus leads to the well-known symptom in Class II, the infraocclusion of the molars and bicuspids. The failure of the mandibular ramus to grow to its normal height restricts the space between upper and lower jaws and, thus, the eruption of upper and lower teeth.

In a case of a true Class III malocclusion, the mandible is too long. Here, cartilagenous growth in the condyle is, for no known reason, accelerated. This entails not only a lengthening of the mandible but also an abnormal heightening of its ramus. Here, the appositional growth of bone, at the posterior border of

the ramus, is outdistanced by the accelerated cartilagenous growth of the condyle and, as a result, the mandible of a true Class III has a long but narrow ramus and an obtuse angle. In these cases we see also an accelerated eruption of teeth because the space into which the teeth erupt is opened at an abnormally great rate of speed due to the accelerated condylar growth.

It is well known that the symptoms of Class III are magnified to the grotesque in some cases of acromegaly. Here too, the syndrome of changes of the mandible has to be realized, that is, the pathologic increase of the mandible in over-all length, the pathologic increase of the mandibular ramus in height, the narrowness of the ramus, the distorted obtuseness of the mandibular angle, and the over-eruption of the teeth.

We have, up to this point, allowed for an analogy between the hyaline cartilage in the mandibular condyle, and the hyaline cartilage of the epiphyseal plate of the long bone. Such an analogy, however, does not exist. To understand the difference between the condylar cartilage of the mandible and the epiphyseal cartilage of a long bone, we must realize that cartilage may grow in two ways: by expansion i.e. by interstitial growth, and by addition i.e. by appositional growth. Interstitial growth of cartilage takes place by mitotic division of chondrocytes which, then, produce new intercellular substance, spreading the cartilagenous substance from within. Appositional growth of cartilage occurs on such surfaces of the cartilage which are covered by connective tissue, perichondrium. Here, the cells in the deepest layers of the connective tissue gradually differentiate into chondroblasts and, then, start to produce hyaline intercellular substance, and so change into chondrocytes. At the border between hyaline cartilage and the connective tissue of its perichondrium, this transitional zone between connective tissue and cartilage can be seen clearly as long as the cartilage grows. Whereas interstitial growth may enlarge cartilage in all directions of space, appositional growth can occur, of course, only on those surfaces which are covered by connective tissue. It contributes, therefore, to the growth of cartilage in certain specific directions. In most cartilages there is a division of labor between interstitial and appositional growth. A cartilagenous epiphyseal plate of a long bone is interposed between the bony diaphysis and epiphysis. In its periphery it is surrounded by perichondrium which is continuous with the periosteum of shaft and extremity. In the direction of the long axis of the long bone, this cartilage grows only interstitially. Its growth in the transverse diameter of the bone, however, takes place only by apposition. In contrast to this type of growth of an epiphyseal cartilage, the condylar cartilage of the mandible grows in all dimensions, at least partly, by apposition. This is due to the fact that the cartilage is bordered by bone only on its deep surface whereas the other surface is covered by fibrous tissue. In fact, histologic sections through the condyle of a young individual show signs of both, interstitial and appositional growth of the condylar cartilage (Fig. 1). Interstitial growth is expressed in the arrangement of the chondrocytes in the deep layers, in columns similar to those which are so characteristic for the zone of proliferation in an epiphyseal cartilage. The border zone between cartilage and the covering fibrous tissue, on the other hand, shows the transition of fibrous tissue into cartilage, and of

fibrocytes to chondrocytes, which is characteristic for the appositional growth of cartilage.

This unique behavior of the condylar cartilage is the reason for the unique behavior of the mandible under certain pathologic conditions. In chondrodystrophic or achondroplastic dwarfs, the interstitial growth of cartilage is severely inhibited by some genetic mutation. However, appositional growth of cartilage, in other words, differentiation of connective tissue into cartilage, is not impaired. It is evident that the long bones of a dwarf of this type remain short but attain a relatively considerable thickness. In the epiphyses of long bones, for instance in the head of humerus and femur, the inhibition of the interstitial growth of the cartilage leads to the formation of characteristic mushroom-shaped extremities of these bones. The articular cartilage does not grow sufficiently in thickness and, thus does not contribute enough to the longitudinal diameter of the developing head. It increases, however, almost normally in circumference because it grows, at its borders, by appositional growth. Here, connective tissue continuing into the articular capsule, covers a narrow marginal area of the cartilage. The consequence of this break in the correlation between interstitial and appositional growth of the articular cartilage is the formation of flat but wide articular ends.

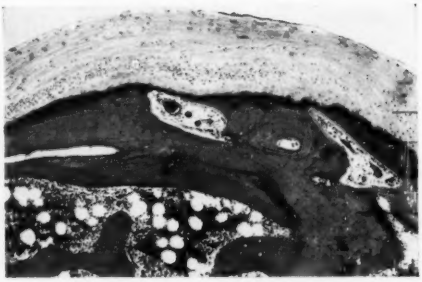

Fig. 6. Section through mandibular condyle of adult, showing cessation of endochondral growth. Hyaline cartilage has disappeared.
a Fibrous covering in mandibular head. In the deep layer isolated chondrocytes.
b Calcified layer.
c Bone.

In the skull, the inhibition of the interstitial growth of cartilage leads to a shortening of the antero-posterior diameter of the cranial base, the deep saddling of the nose and the prominence of the forehead. It is interesting that the harmony between upper and lower jaws is, generally, not disturbed although the mandible grows in length and height by the cartilagenous growth in the condyle. This peculiar behavior of the mandible, in such cases, can be easily understood if it is remembered that the condylar cartilage of the mandible may and does grow interstitially *and* by apposition under normal conditions. If interstitial growth is inhibited, as in achondroplasia, appositional growth can fully compensate for the lack of appositional growth so that, in the end, the over-all growth of the mandible is not impaired. The result is even sometimes a mandibular protrusion as is the rule in the chondrodystrophic races of dogs, *e.g.* bulldogs.

The overgrowth of the mandible, which is so characteristic for many cases of acromegaly, can also be explained by the peculiar structure of the condyle. There is, first, the fact to be considered that the hyaline cartilage of the condyle persists to a higher age than any epiphyseal plate in any part of our body. An overproduction of growth hormone by a tumor of the hypophysis may, therefore, stimulate the growth of the cartilage at an age when other bones have already lost their capacity of longitudinal growth. But even in a case in which the hypophyseal tumor develops at an age when the hyaline cartilage

in the mandibular head has already disappeared, the mandible may still respond to an increased growth stimulus. The reason for this is the covering of fibrous tissue on the articular end of the mandible (Fig. 6) which might proliferate in its deep layers and again differentiate into cartilage. This newly formed cartilage may then again, by its proliferation, initiate a growth of the mandible in length and height. That this re-awakening of the growth potency of the mandibular condyle will lead to a characteristic shape of the over-enlarged mandible has been mentioned before.

In the preceding pages an attempt has been made to correlate the findings on structure and growth pattern of the mandible to certain physiologic and pathologic conditions. The conclusions should not be regarded as final, rather as a stimulus for further discussion.

REFERENCES

1. Brodie, Allan G.: On the Growth Pattern of the Human Head from the Third Month to the Eighth Year of Life. *Am. J. Anat. 68*:209, 1941.
2. Toldt, Carl: Ueber das Wachstum des Unterkiefers. Z. *Heilkunde*, 5:1, 1884.
3. Diamond, Moses: The Patterns of Growth and Development of the Human Teeth and Jaws. *J. D. Res. 23*:273, 1944.
4. Sicher, Harry and Weinmann, Joseph P.: Bone Growth and Physiologic Tooth Movement. *Am. J. Orthodont, and Oral Surg.* Vol 30, *Oral Surgery* p. 109, 1944.
5. Weinmann, Joseph P. and Schour, Isaac: Experimental Studies in Calcification. II. The effect of a Rachitogenic Diet on the Alveolar Bone of the White Rat. *Am. J. Path.* (in print).
6. Orban, Balint: *Oral Histology and Embryology*. Chapters VIII and XIV. St. Louis, Mosby, 1944.

There will be no annual meeting of the American Academy of Periodontology during 1945 because of travel restrictions.

Bone Changes in the Jaw Caused By Renal Hyperparathyroidism

By JOSEPH P. WEINMANN, M.D.* *Chicago, Illinois*

THE OBJECT of this report is to describe changes in the jaws which are attributed to a hypertrophy of the parathyroid glands secondary to a chronic insufficiency of the kidneys. Although skeletal changes in renal hyperparathyroidism have been observed (1,2,5,6,7,16) there are no reports available on similar changes in the jaws.

MATERIAL

The findings are based on histologic and roentgenographic investigations of three upper and lower jaws.

Case I: A white male who died of glomerulonephritis and uremia at the age of 64.

Case II: A white male who died of chronic glomerulonephritis and hypertension at the age of 51.

Case III: A colored male who died of lobar pneumonia and glomerulonephritis at the age of 32. The patient had rheumatic fever at the age of 24 and, at the time of his fatal illness, he suffered from an acute arthritis of the right elbow and knee. The osteitic changes in the lower jaw of this case were reported by R. Kronfeld (12) and diagnosed as osteitis similar to Paget's disease.

In all three cases the upper and lower jaws were photographed and radiographed prior to histologic preparation. The jaws were separated in blocks of two to three teeth, sectioned in different planes and stained with hematoxylin-eosin.

Fig. 1. Case I. Mesio-distal section through right upper first and second molars. Focus of osteitis fibrosa cystica (a) in the floor of the maxillary sinus, the lining of which shows chronic inflammation.

FINDINGS

Case I. Osteitic changes are found histologically but not roentgenographically in the molar region of both sides of the upper jaw. The foci are situated in the bone separating the roots of the second right and left molars from the maxillary sinus (Fig. 1). The periodontal membrane reveals no inflammatory changes but an exudate with lymphocytes and plasma cells is present in the maxillary sinus. The bone marrow spaces are enlarged and filled with fibrous marrow (Figs. 2 and 3) but have no signs of inflammation. The fibrous marrow consists of a loose and partly edematous connective tissue. The

* Department of Research, Loyola University Dental School, Chicago College of Dental Surgery. Presented at the Postgraduate Course of the American Academy of Periodontology, W. K. Kellogg Foundation, Ann Arbor, Michigan, May, 1945.

cells are numerous and have large round nuclei. Multi- and uni-nuclear osteoclasts are found in Howship's lacunae indicating active resorption. Some giant cells are found free in the fibrous tissue. In other areas of the same bone marrow spaces bone apposition is indicated by the presence of osteoblasts and a thin layer of osteoid tissue. In other areas of the same bone marrow spaces bone apposition is indicated by the presence of osteoblasts and a thin layer of osteoid tissue. In one large bone marrow space cysts are seen (Fig. 2) filled with serum. They do not have an independent wall although the connective tissue of the marrow space is slightly condensed in some sectors of the periphery of the cysts. Edematous infiltration of other areas of the marrow space can be seen in the adjacent zones and at some distance to the cysts. The bone marrow shows dilated vessels. There are no signs of fresh or old hemorrhages in or around the cysts. The medium sized blood vessels in the periosteum covering the

Fig. 2. Case I. Higher magnification of area (a) in Fig. 1. Resorption of bone. Fibrous marrow contains giant cells (b) and cysts (a) filled with serum.

Fig. 3. Case I. Focus in the floor of the left maxillary sinus. Lining of sinus (c) shows signs of chronic inflammation. Marrow spaces filled with fibrous marrow. Resorption at a; apposition at b. Periapical connective tissue at d.

maxilla show calcification (Fig. 4A) of the tunica media. The periosteum contains some calcified bodies possibly being obliterated and completely calcified small arteries in cross section (Fig. 4B).

Case II. Two foci of osteitis were found, one in the bony scar after extraction of the left lower central incisor (Figs. 5 and 6), and one adjacent to the apex of the upper left central incisor (Fig. 7).

The alveolar ridge at the site of the extracted lower left central incisor is covered by a thick layer of dense connective tissue without any signs of inflammation. The stratified squamous epithelium in this area is hornified. The alveolar bone itself is rarefied in the space between the right central and left lateral incisors. In the roentgenogram and histologic sections (Figs. 5A and B) this rarefaction extends beyond the limits of the alveolus of the extracted tooth, from the lamina dura of the right central to that of the left lateral incisor. In the microscopic section the surface of the alveolar process at the site of extraction is formed

by a bony plate consisting of lamellated bone. The plate (Fig. 6) is in the center of its deep surface thinned out by still progressing resorption. Near the surface a small area of the bone shows the changes characteristic of osteitis fibrosa. This focus consists of irregularly shaped and arranged bone trabeculae. Only a few trabeculae consist of mature lamellated bone. The rest is composed of coarse fibrillar bone of varying degrees of immaturity. Osteoblastic and osteoclastic activity can be observed in several places. The marrow consists of rather dense connective tissue which is fairly rich in cells, with spindle shaped, dark staining nuclei. Nowhere are any signs of inflammation.

The second focus not visible in the roentgenogram is found in the immediate vicinity of the alveolar fundus of the left upper central incisor (Fig. 7A). The pulp of this tooth is necrotic and the periapical tissue shows slight inflammatory infiltration. The upper right central incisor had sustained a fracture which had healed (11). The pulp necrosis of the left central incisor was probably caused by the same trauma. The focus above the root of the upper left central incisor (Fig. 7B) is characterized by

Fig. 4. Case I. A: Calcification (a) of tunica media in periosteal arteries. B: Calcified bodies (b) in periosteum.

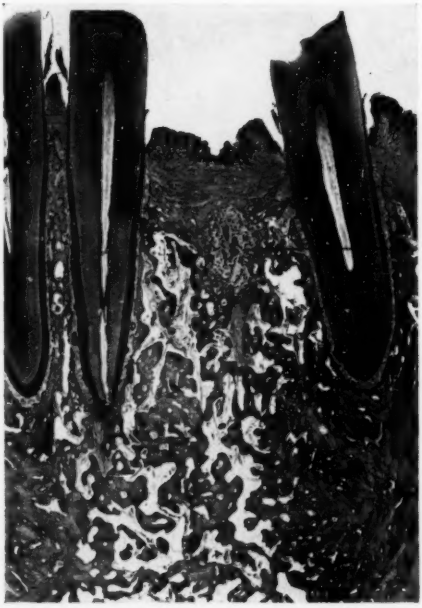

Fig. 5. Case II. A: Roentgenogram of lower incisor region. Osteoporotic area between right central and left lateral incisors. B: Mesio-distal section through the focus (a).

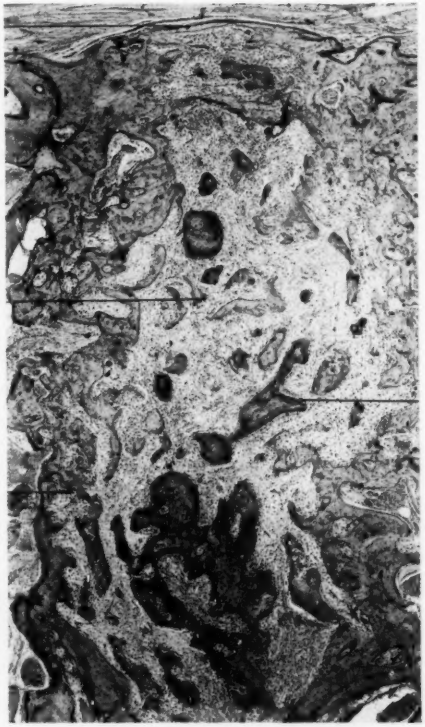

Fig. 6. Case II. High magnification of the focus of Fig. 5. Lamellated bone on the surface (a). Fibrous marrow (b). Bone apposition at d, bone resorption at c.

extensive osteoclastic resorption which had hollowed out some thick trabeculae of the densely arranged spongiosa of this area. Some of the trabeculae are split by this undermining resorption. The newly-formed marrow spaces replacing the core of the affected trabeculae are filled with fibrous loose connective tissue. The density of the connective tissue is approximately intermediate between that of the loose and edematous tissue in Case I and that of the dense connective tissue of the focus in the lower jaw of Case II. The neighboring marrow spaces of this region, contain fatty marrow. On the surface of the bony trabeculae which is directed toward the non-affected marrow spaces active bone apposition is in progress, compensating for the weakening of the trabeculae by the dissecant resorption.

Case III. In the maxilla a minute focus can be found in the floor of the right maxillary sinus above the root of the first molar. This focus (Fig. 8), however, was too small to be seen in the roentgenogram. The lamellated bone in the floor of the maxillary sinus is partly destroyed by active osteoclasis. There are signs of compensatory apposition in some areas. In parts the old lamellated bone has been replaced by coarse fibrillar bone. The marrow in the focus is fibrous and consists of rather loose connective tissue. It is noteworthy that the lining of the maxillary sinus is chronically inflamed and an inflammatory polyp has developed in the region of the osteitic focus.

Four more foci of localized osteitis fibrosa are present in the mandible. They could be distinguished in the roentgenograms (Fig. 9) and are situated in the neighborhood of the root apices of the right and left lower third molars and in the apical region of the right and left lower first molars.

All mandibular foci have certain characteristics in common (Figs. 10-14). They have developed in the spongy bone; a great amount of bone trabeculae of the mature lamellated type has been destroyed by osteoclasis which, in some places, is still in progress (Fig. 11B). The defect in the bone is filled with an irregular spongy bone, the trabeculae of which are thin and short. The meshwork is in some areas very dense, in others the trabeculae are separated by large marrow spaces. The bone in the foci is of the primitive, immature or coarse fibrillar type. The degree of its primitiveness varies considerably. The marrow spaces contain nowhere normal fatty or cellular marrow but a rather dense fibrous connective tissue.

The focus in the apical region of the right lower first molar (Fig. 11A), of which only small root remnants are present, consists of three densely arranged single foci which are separated from each other by narrow layers of normal spongy bone. The most mesial of these foci reaches into the region of the second bicuspid (Fig. 12). At this level the labial cortical plate has been resorbed in a small area and the focus reaches to and protrudes slightly over the periosteal surface of the mandible.

Some of the foci contain in their center irregularly rounded masses of lamellated compact bone (Figs. 11 and 13). There is little doubt that this is bone which escaped destruction during the development of the

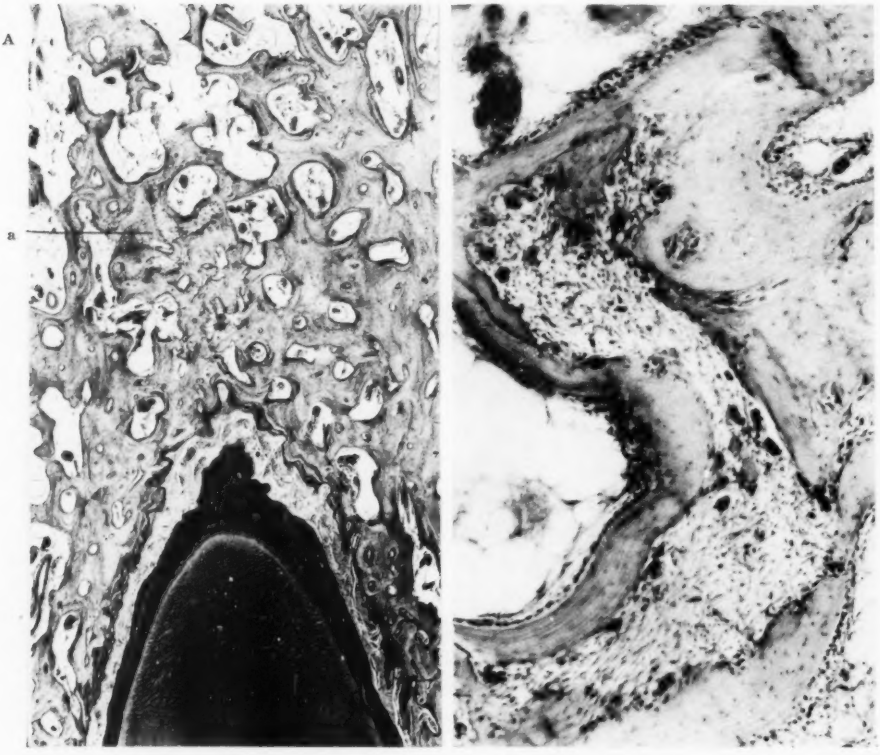

Fig. 7. Case II. A: Focus (a) in the apical area of left upper central incisor. B: Detail in higher magnification. Splitting of a trabecula by undermining resorption. Core of the trabecula replaced by fibrous marrow containing giant cells. Apposition on the surface of the trabeculae bordering the marrow spaces filled with fatty marrow.

foci. It is highly probable that this compact bone is connected with the cortical compacta.

The correlation of the histologic and roentgenologic findings is of great practical importance. The most striking feature is the fact that these foci are visible in the roentgenogram as areas of increased radiolucency. Their constituent tissue, immature coarse fibrillar bone, is far less radiopaque than mature lamellated bone. Even an osteosclerotic area consisting of primitive bone is less radiopaque than normal lamellated spongy bone.

DISCUSSION

Pathogenesis. The described changes warrant a diagnosis of osteitis fibrosa. The changes consist mainly of a more or less extensive resorption of mature lamellated bone and its replacement by spongy bone of the immature coarse fibrillar type and fibrosis of the bone marrow in these foci. The history of the presented cases gives no indication of a generalized osteitis fibrosa. The foci are situated in areas that are either traumatized or damaged by an adjacent infection. One of the four "traumatic" foci is found in the bony scar of an extraction; a second in the apical region of a tooth, the neighbor of which had sustained a root fracture; two foci are found at the roots of two lower third molars which are in horizontal position and under occlusal trauma. Three of the five "inflammatory" foci are situated in the floor of a chronically infected maxillary sinus and two in the apical region of the infected lower first molars.

Hyperparathyroidism which causes osteitis fibrosa may be primary or secondary. Primary hyperparathyroidism is caused by a

Fig. 8. Case III. Focus of osteitis fibrosa in the bone between right upper first molar (a) and maxillary sinus (b). Resorption of bone at c, apposition at d. Fibrous marrow (e).

tumor of one or more of the parathyroid glands. Secondary hyperparathyroidism is in many cases due to a hypertrophy of the glands induced by renal insufficiency (3, 14, 15). The renal pathology in the three described cases is, therefore, of great significance.

The mechanism of parathyroid hypertrophy in renal insufficiency is explained in the following way: Normally, the parathyroids function to maintain the optimal calcium blood level by regulating the mobilization of calcium from the skeleton. Since there is no decalcification of living bone, in other words, no extraction of calcium salts from the living bone, the mobilization of calcium entails osteoclastic resorption of the bone. One function of the parathyroid gland, can, therefore, be described as regulating the differentiation and activity of osteoclasts. The mobilization of calcium by osteoclasis is necessarily and inseparably link-

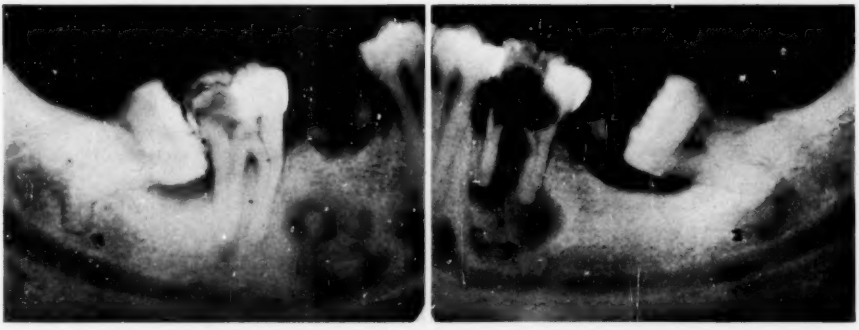

Fig. 9. Case III. Roentgenogram of right and left lower molar regions.
Focus x see Fig. 10
Focus 000 see Figs. 11 and 12
Focus Y see Fig. 13
Focus Z see Fig. 14

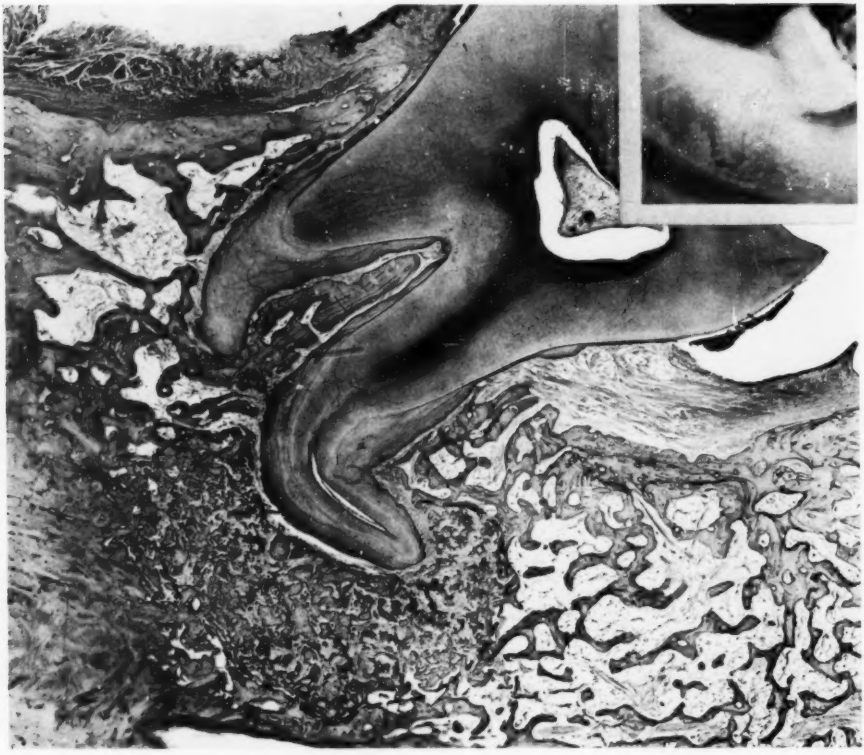

Fig. 10. Case III. Focus (x of Fig. 9) in the apical region of right lower third molar indicated by broken line.

ed with the mobilization of phosphorus. The excess phosphorus is eliminated by the second main function of the parathyroid glands, that is, by its direct influence upon the kidneys. Here, it exerts an inhibitory influence on the reabsorption of phosphorus in the tubules and thus, achieves an increase in phosphorus excretion.

The organism reacts to a rise in the phosphorus blood level, which may occur under different conditions, by a hyperactivity and finally hypertrophy of the parathyroids. Such a secondary hypertrophy of the parathyroids can be produced experimentally by continued injection of phosphates (4). The same effect is achieved in all cases of renal insufficiency in which a phosphorus retention leads to a rise of the phosphorus blood level. Since the hyperactivity of the parathyroid gland cannot result in an adequate increase of phosphorus excretion because of the renal damage, a vicious circle arises. After a time the excess parathyroid hormone will produce changes in the skeleton because the two functions of the parathyroid gland, that is the influence on the kidneys and the stimulation of osteoclastic activity, cannot be separated. In cases of severe renal damage the typical picture of generalized osteitis fibrosa develops.

Hyperparathyroidism does not attack all bones or all areas of one bone to the same degree (9, 18). Osteoclasis seems to be most intense in areas where bone growth, that is, a combination of osteoblastic and osteoclastic activity, is most pronounced. It may be assumed that areas of increased osteoclastic activity after any injury to a bone, will likewise be the first where any increase of parathyroid activity may become manifest. This would lead to a concept of a latent hyperparathyroidism. It may be defined as a hypersecretion of the parathyroids, pri-

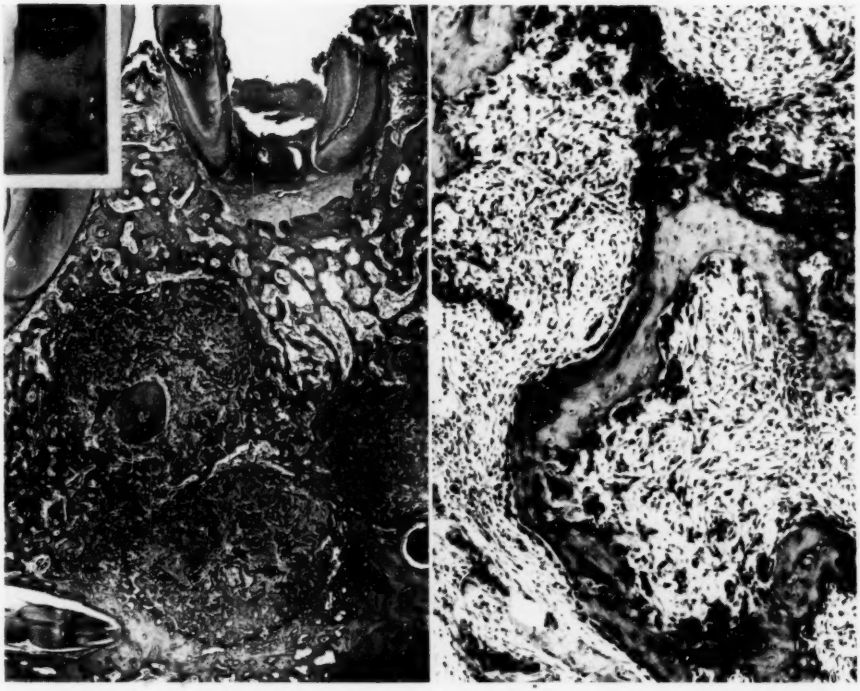

Fig. 11. Case III. A: Three areas of focus (000 of Fig. 9) in the apical region of the right lower first molar indicated by broken lines. B: Detail from one of the foci showing the interplay between bone resorption and bone apposition.

mary or secondary, which, in the relatively resting adult skeleton, does not produce pathologic changes. In a case of local injury, however, and therefore a heightened local reconstructive activity in a bone, this focus would be a locus minoris resistentiae at which the heretofore latent hyperparathyroidism would lead to a pathologic exaggeration of the skeletal reaction. As in generalized osteitis fibrosa, a far-reaching osteoclastic destruction of bone, its more or less complete replacement by immature coarse fibrillar bone and fibrosis of the bone marrow may be anticipated. As in generalized osteitis fibrosa, cysts may form which are characterized by their lack of a specific lining. Metastatic calcification in kidneys and blood vessels are frequent.

CORRELATION OF ROENTGENOGRAPHIC AND HISTOLOGIC FINDINGS

It is especially significant that the resorbed mature bone is replaced by coarse fibrillar or immature or primitive bone. The bone formed in early stages of development is always of this primitive type and always spongy bone. Normally, this immature bone is gradually replaced by mature and lamellated bone. In the adult, bone of the primitive type is formed under varying conditions, the common factor being the rapid apposition of bone. Thus, we see formation of immature bone in callus formation after fractures; as osteophytes in what may be called emergency reinforcement; and finally in bone-forming tumors. Primitive bone is characterized by a large amount of coarse, irregularly arranged bundles of fibrils in the ground substance, by the irregular arrangement of the rather large and rounded osteocytes which possess relatively few processes and, finally, by its incomplete calcification. The fact that this primitive bone is far less calcified than mature bone renders it more radiolucent than mature bone. This is the reason why, for instance, the early *bony* callus cannot be detected in a roentgenogram although histologic investigation reveals far advanced

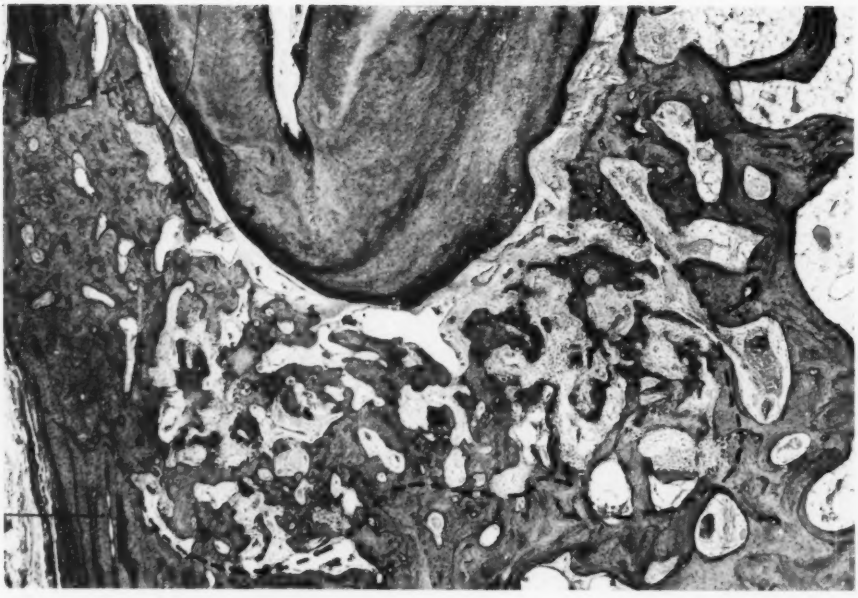

Fig. 12. Case III. Bucco-lingual section through mesial area of the focus of Fig. 11A. indicated by broken line at the apex of right lower second bicuspid. A: The compact of cortical layer of the mandible partly replaced by the focus.

bone production. It is important to realize that the roentgenogram hardly permits differentiation of primitive bone from soft tissues as cartilage, muscles, and so forth.

Foci of localized osteitis fibrosa appear, therefore, in the roentgenogram as defects of the bone. Only a close comparison with actual defects of bone (Fig. 8) permits a differentiation on the roentgenogram because an osteitic focus is slightly less radiolucent than a defect caused by a radicular cyst or granuloma. A further means of differentiation is the cloudy or spotty appearance of an osteitic focus. The presence of a dense central area equaling in density that of normal bone would also strengthen the diagnosis of osteitis fibrosa. Such central areas of normal bone are, however, found only occasionally and only in the lower jaw.

The correct diagnosis of a round radiolucent area in the jaw will meet greater difficulties if such a focus is situated around or adjacent to the apex of a tooth or is projected upon the apex by roentgenography. It is possible that some radiolucent areas on the root of a tooth with a vital pulp, diagnosed as granuloma or cyst, were foci of localized osteitis fibrosa.

A practical evaluation of the findings is twofold. It becomes apparent that root canal treatment or extraction of a vital

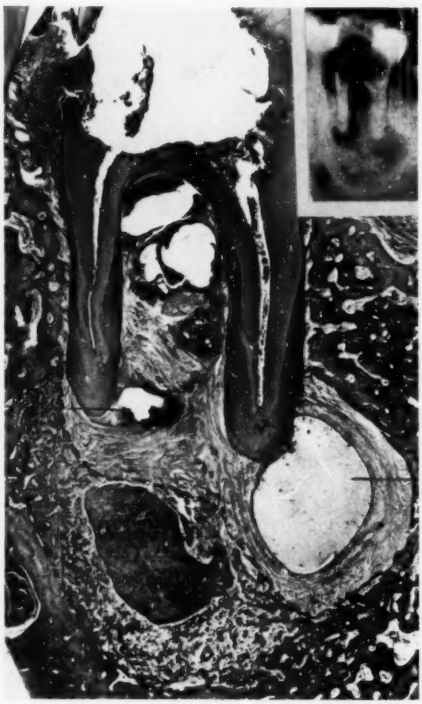

Fig 13. Case III. Focus (Y of Fig. 9) in the apical region of the left lower second molar indicated by broken line. a=radicular cyst on distal root. b=epithelial granuloma on mesial root.

Fig. 14. Case III. Focus (Z in Fig. 9) in apical region of left lower third molar indicated by broken line.

tooth connected with an osteitic focus is not only of no therapeutic value but may even lead to a deterioration by causing a new trauma. Stafne and Austin as well as Kronfeld already warned of surgical interference in similar cases. In most cases secondary hyperparathyroidism in the adult is of renal origin. The knowledge of a renal disturbance previously diagnosed will do much to strengthen our diagnosis of a radiolucent area as osteitic focus. On the other hand, the possibility of this etiology in a case of bony defect in the jaws which cannot be easily accounted for, may lead to an early diagnosis of a hitherto undiagnosed renal damage.

SUMMARY

1—The foregoing report is based on the roentgenologic and histologic examination of teeth and jaws of three individuals suffering from renal insufficiency.

2—In all of these jaws foci were found in which the mature bone had been destroyed by osteoclasis and replaced by immature coarse fibrillar bone. The marrow in these foci was fibrous.

3—The smallest of these foci could not be seen in the roentgenogram; the larger foci appear in the roentgenograms as radiolucent areas.

4—The lesions can be diagnosed as secondary localized osteitis fibrosa.

5—The concept of a latent hyperparathyroidism is advanced. According to this concept hyperparathyroid foci develop only in such localization where trauma or infection led to increased reconstructive activity of bone.

6—The practical significance of the radiolucency of primitive coarse fibrillar bone is discussed.

7—The development of a granuloma or a cyst on the root of a tooth with vital pulp seems more than ever questionable. Such foci are, in all probability, localized osteitis fibrosa.

BIBLIOGRAPHY

1. Albright, F., Drake, T. G., and Sulkowitsch, H. W.: Renal Osteitis Fibrosa Cystica. *Bul. Johns Hopkins Hosp.* 60:377, 1937.
2. Anderson, W. A. D.: Hyperparathyroidism and Renal Disease. *Arch. Path.* 27:753, 1939.
3. Guguid, J. B.: Hyperparathyroidism in Experimental Nephritis. *J. Path & Bact.* 54:177, 1942.
4. Eger, W.: Weitere Untersuchungen Zur Experimentellen Osteitis Fibrosa. *Virchow's Archiv.* 306:183, 1940.
5. Follis, R. H., Jr., and Jackson, D. A.: Renal Osteomalacia and Osteitis Fibrosa in Adults. *Bul. Johns Hopkins Hosp.* 72:232, 1943.
6. Gall, E. A., and Bennett, G. A.: Osteochondritis Deformans of Hips and Renal Osteitis Fibrosa Cystica. *Arch. Path.* 33:866, 1942.
7. Ginzler, A. M., and Jaffe, H. L.: Osseous Findings in Chronic Renal Insufficiency in Adults. *Am. J. Path.* 17:293, 1941.
8. Haselhofer, L., and Bauer, W.: Beitrag Zur Frage der Braunen Tumoren und der Oertlichen Osteitis Fibrosa des Unterkiefers. *Vtschr. F. Zahnheilk*, 46:487, 1930.
9. Jaffe, H. L., Bodansky, A., and Blair, J. E.: Sites of Decalcification of Bone Lesions in Experimental Hyperparathyroidism. *Arch. Path.* 12:715, 1931.
10. Ingalls, T. H., Donaldson, G. A., and Albright, F.: Locus of Action of Parathyroid Hormone: Experimental Studies with Parathyroid Extract on the Normal and Nephrectomized Rat. *J. Clin. Investigation.* 22:603, 1943.
11. Kronfeld, R.: A Case of Tooth Fracture, with Special Emphasis on Tissue Repair and Adaptation Following Traumatic Injury. *J. D. Res.* 15:429, Dec., 1936.
12. Kronfeld, R.: Die Diagnostische und Pathologische Bedeutung Ostischer Herde im Menschlichen Kiefer. *Ztschr. F. Stomat.* 35:177, 1937.
13. McJunkin, F. A., Tweedy, W. R., and McNamara, E. W.: Effect of Parathyroid Extract and Calciferol on the Tissues of the Nephrectomized Rat. *Am. J. Path.* 13:325, 1937.
14. Pappenheimer, A. M., and Willens, S. L.: Enlargement of the Parathyroid Glands in Renal Disease. *Am. J. Path.* 11:73, 1935.
15. Pappenheimer, A. M.: The Effect of Experimental Reduction of Kidney Substance upon the Parathyroid Glands and Skeletal Tissue. *J. Exper. Med.* 64:965, 1936.
16. Park, E. A., and Eliot, M. M.: Renal Hyperparathyroidism with Osteoporosis (Osteitis) Fibrosa Cystica in Brennemann. *J. Practice of Pediatrics.* Hagerstown, Md. W. F. Prior Co., Inc. Vol. 3, Chapter 29, 1937.
17. Stafne, C. E., and Austin, L. T.: A Study of Dental Roentgenograms in Cases of Paget's Disease (Osteitis Deformans), Osteitis Fibrosa Cystica and Osteoma. *J.A.D.A. and D. Comos.* 25:1202, August, 1938.
18. Weinmann, J. P., and Schour, I.: Experimental Studies in Calcification. III. The Effect of Parathyroid Hormone on the Alveolar Bone and Teeth of the Normal and Rachitic Rat. *Am. J. Path.* (In Press).

BOOKS RECEIVED

Periodontal Diseases and Soft Tissue Lesions of the Oral Cavity. Diagnosis and Treatment. By Arthur H. Merritt, M.S., D.D.S., Sc. D., F.A.A.P. (New York, the Macmillan Company, 1945.) 258 pp., 53 illustrations. *To be reviewed* in the October issue of this Journal.

Dental Chronology. A Record of the More Important Historic Events in the Evolution of Dentistry. *By* Herman Prinz, A.M., D.D.S., M.D., Sc.D., Dr. Med. Dent. (Philadelphia, Lea & Febiger, 1945.) 189 pp. Illustrated with 97 engravings. *To be reviewed* in the October issue of this Journal.

Teaching in Colleges and Universities with Special Reference to Dentistry. By Lloyd E. Blauch and Associates. This book is now available and may be obtained from the Business Manager, *Journal of Dental Education,* Maynard K. Hine, 1121 West Michigan Street, Indianapolis 2, Indiana. This valuable book of 349 pp. deals broadly with teaching at the University and College level.

Nutrition and Dental Health

III. VITAMIN B-COMPLEX

By DOROTHEA F. RADUSCH, D.D.S., B.S., M.A., *Minneapolis 2, Minnesota*

THE DIETARY standards as outlined previously and based on the Table of Recommended Allowances as given by the Food and Nutrition Board of the National Research Council* were intended as a guide for good dietary planning in civilian life and in the armed services. The standards are applicable to the entire population. They are liberal enough to provide for those whose requirements are higher than the average.

It has been established that many adult persons can subsist in apparent good health on less than half the amount of some of the items listed. One such example is protein intake. But the individual has no way of knowing whether his needs are below average or above it. Most certainly, lack of symptoms of recognized deficiency disease is insufficient evidence of adequacy.

Then too, it may be pertinent to the present discussion, to point out that Ivy *(Quart. Bul. N.W.U. Med. School* 18:22, 1944) in discussing the question of normality states that the "clinical" view of normality is one which "views the mild imperfections of the body as normal".

A further deterrent to good nutrition is the complacency and smugness expressed in the frequently heard comment, "We always try to set a good table."

The good table, it should be noted, does not mean nutritionally sound meals. On the contrary, it means meals from which the most nutritious foods have been refined, peeled and trimmed away: meals of white bread and polished rice and refined sugar—with the food values largely discarded in the refining process; meals of white celery, blanched lettuce, and beet roots, with the good outer and top leaves thrown away; meals of cabbage and asparagus and potatoes—all trimmed and peeled of their most vitamin-rich parts; meals of meat shrunk by being cooked at too high a temperature, and with the fatty parts left to be trimmed away on the plates. This is the "good table" concept that keeps the amount of garbage pretty uniform, no matter from what part of town it is collected. (Dr. Robert Oliver, *American Scientist* 32:271. Oct. 1944.)

To assure a liberal supply of essential nutrients, the meaning of the "good table" should become more closely identified with that of the food-pattern suggested and based upon the recommendations of the National Research Council.

EVALUATION OF EXPERIMENTS ON HUMAN SUBJECTS

There have been objections by some scientific workers that certain standards were set too high according to results of recent experiments. This evidence, as pointed out by Wilder *(Science* 101:288, Mar. 23, 1945),

. . . Although representing carefully conducted investigation, all relates to small groups of experimental subjects studied for periods—at the longest six to nine months—representing no more than 1 per cent of the life span of the human species.

Typical of such experiments are those of Keys and co-workers *(J. Nutrition* 27:485, 1944) abstracted and discussed in *Nutrition Reviews* 2:335, Nov. 1944. The essentials of this report are repeated here:

In this instance, 8 normal young men were studied to observe the effect of reduction of intake of B-complex vitamins on physical efficiency. The 8 men were fed a highly

* *See J. Periodont.* 16(2):52. Apr. 1945.

restricted diet consisting of such materials as macaroni, polished rice, bread and butter, jelly and a special cream made by diluting a preparation of 70 per cent butterfat. Small amounts of canned fruits were provided. All flours and cereals were low-vitamin preparations and only flavoring amounts of chicken, beef or veal were allowed. The daily average protein intake on this diet was 85 g. and roughly 35 per cent of the calories were provided as fat. Supplements of minerals and the fat soluble vitamins were furnished in the amounts recommended by the National Research Council. During the first 21 days, all subjects received the amounts recommended by the Council. For the next 14 days only the deficient basal diet was fed. This supplied 0.16 mg. of thiamine* per 1000 calories, 0.15 mg. of riboflavin, and 1.8 mg. niacin. (Recommended for males 0.6 mg. thiamine and 0.9 mg. riboflavin per 1000 calories.)

Five of the subjects received placebos (non-active substitute capsules) while three received abundant B-vitamins. Neither the subjects nor the observers knew which they were receiving. The subjects performed fixed tasks, most of them on treadmills, involving a daily expenditure of 4500 to 5000 calories. Extensive series of physiologic, biochemical, and psychologic tests were made at intervals. These included measurements of endurance, anaerobic work, speed, coördination, and muscular strength, and questionnaires were filled out periodically. Chemical studies were made of the blood and urine.

As might be expected, the vitamin content of the urine was found to vary with the vitamin intake of the subjects. All other tests failed to show any correlation whatever with vitamin intake; in other words, there was no evidence of physical or psychologic deterioration after 14 days of hard work on a diet very low in the vitamin-B-complex.

If one may generalize from so few cases, perhaps there is indication from this experiment that at least for a few weeks, a normal man can perform severe physical work on a diet relatively low in B-vitamins without evidence of harm. Hulse *et al* (*Ann. Int. Med.* 21:440, 1944) claim that a still lower intake of thiamine, less than 0.2 mg., gave clinical evidence, chiefly a decline in the sense of well-being correctible by thiamine, by the end of the second week. *Nutrition Reviews* 2:328, Nov. 1944 contains a discussion of Dietary Thiamine Levels and Deficiency symptoms in which it is stated that when thiamine was reduced to between 0.17 and 0.12 mg. per 1000 calories the subjects became irritable and easily provoked in less than three weeks. It was also noted, that when experimental and control groups are interchanged, the incidence of favorable change is such that the response is greater to which ever pill (test or placebo) was given first. It would appear that the factor of suggestion is so great that any attempt to use subjective responses to therapy in evaluating the effect of supplements is hazardous, even though a control group is used. Perhaps partly because of this, the many experiments set up by Keys and his co-workers have measured objective responses, primarily muscular or work tests. Their reports as well as those of many others on human nutrition tests do not include data on oral conditions during or following the test periods. One must assume that such examination was not included as part of the elaborate series of tests. From the dental viewpoint, omission of such observations seems most regrettable.

Unfortunately too, human experiments are costly and thus usually performed with small groups, leading to the temptation to draw unwarranted broad conclusions. It is clear that more work needs to be done to establish the limits of abuse, dietary or otherwise, that man can tolerate. Furthermore, it is probable that most examples of full-fledged or even mild deficiency disease result from protracted rather than brief dietary deprivations.

Apropos of this Dr. Wilder said: (*Science* 101:285, Mar. 23, 1945)

Much investigation with lower animals has shown that allowances which seem to be quite adequate to provide for health and vigor for short periods of the life span are quite inadequate to maintain good health for the life span as a whole. Furthermore,

* Thiamine spelled with an "e" is the preferred spelling as decided by the Nomenclature Committee of the American Society of Biological Chemists.

good nutrition must demand that the individual shall possess stores of nutrients as reserves to meet emergencies. There has been dispute about whether evidence of diminishing reserves is acceptable as a criterion of requirements. Be that as it may, the fact remains that persons who possess reserves are better able to withstand not only temporary deprivations but also stresses such as are involved in surgical operations or disease, than persons who lack reserves. This has long been established for protein and iron, and evidence is now at hand that it applies as well, possibly even more acutely, to reserves of vitamins and especially to the body's store of thiamine, which is relatively rapidly depleted when the intake falls below requirements.

On the other hand, the recent evidence suggests that requirements for thiamine and riboflavin are less closely related to the total calories of the diet or to total expenditure of energy than appeared to be the case when allowances for these vitamins were recommended. The present recommendations of 0.6 mg. of thiamine and 0.9 mg. of riboflavin for each 1000 calories are readily defensible when the calorie intake is 2500 or less. They are less defensible for higher calorie brackets. The great majority of diets for adult men and women provide not less than 2000 nor more than 3000 calories. Therefore, it is justifiable to consider setting a single allowance for thiamine (1.5 mg.) and one for riboflavin (2.0 mg.) for all normal adult men and women, with an accompanying suggestion that, when either the calorie value of the diet or expenditure of energy is much more than 2500 or 3000 calories, additional thiamine and riboflavin may be necessary.

DIETARY LACK OF B-COMPLEX VITAMINS

Even though vitamin deficiencies may not arise only because of an incomplete diet, but may arise presumably because of some metabolic disturbance, lack of intake is the more usual reason for deficiency. According to Wilder *(Ann. Int. Med.* 14:2189, 1942) and Lane, Johnson, and Williams *(J. Nutrition* 23:613, 1942) mild forms of thiamine deficiency are rather widespread because of want of thiamine in the American diet. The introduction of enriched flour and bread, as a temporary war measure has increased the average intake of thiamine from 0.8 mg. per 2500 calories to 1.3 mg. This nearly meets the requirements of the average person, except during periods of stress or special conditioning.

Such special conditioning may be brought about for example, by the use of sulfonamide compounds. Use of these may increase the need for thiamine by destruction of bacteria in the bowel which normally, by biosynthesis, apparently supply part of the body's vitamins *(J.A.M.A.* 123:683, 1943). Or, such therapeutics may interfere with utilization of vitamins through competitive blocking of receptor radicles in enzyme systems which for normal functioning are occupied by vitamins.

Furthermore, the more starches or sugar eaten, the greater the need for the B-vitamins. The more one dilutes these vitamins with calories from pure sugar, the worse the nutritional status. Those who habitually eat candy or sweetened carbonated drinks between meals, increase their need for B-vitamins. Unfortunately, by those same practices, they often substantially reduce the amount of more nourishing food they should be eating which would supply the necessary vitamins.

Between meal eating may be useful for relieving fatigue and increasing productivity, but it should contribute to and not detract from the total daily nourishment.

Dr. Roger Williams has stated (p.264, Wohl, *Dietotherapy;* Saunders Co. Phil. 1945) that, "because of the fundamental nature of the B-vitamins for

every cell in the body, their deficiency may present problems not encountered in those vitamins which may function primarily in a localized area or tissue. What is termed a 'generalized deficiency' may develop. Every tissue in the body may suffer before any particular tissue breaks down sufficiently to cause observable pathologic change." Such a deficiency is more difficult to recognize, but may be none the less real.

SYMPTOMS OF B-VITAMIN LACK

Beri-beri and pellagra, diseases commonly thought of as due primarily to deficiency in a single vitamin are actually multiple-deficiency conditions in which one deficiency is more acute than others. The dramatic and prompt healing of oral lesions in the treatment of human pellagra has indicated a possible relationship between the various B-complex factors and oral diseases, especially of the Vincent's infection type. The treatment of pellagra with nicotinic acid* brings about healing of ulcers beneath the tongue and on the lips within three to five days. There is marked relief of soreness and pain in the mouth while the papillae of the tongue regenerate within 7 to 14 days. The dosage used is 50-100 mg. given orally three to five times a day.

The descriptions of ariboflavinosis state that it is characterized by cheilosis which usually appears as a transverse fissure in each angle of the mouth with varying degrees of denudation, reddening, and scaliness of lips. The lesion begins as a grayness of the mucosa at the angles of the mouth, and this is followed by maceration and fissuring. The fissures are superficial and do not bleed, but may be covered with a yellowish dried exudate. Changes in the lips other than at the angles of the mouth may be very mild. The tongue appears to be smooth, due to changes in the papillae, and the entire tongue may have a purplish tinge.

Sydenstricker *(Am. J. Pub. Health* 31:344, April, 1941) says that soreness of the tongue is a characteristic of riboflavin deficiency. Sebrell and Butler *(Pub. Health Rep.* 53:2282, Dec., 1938) fed 18 women a diet deficient in riboflavin. Ten quickly developed lesions of the lips. In all of these cases, the lesions disappeared when 1-2 mg. of pure riboflavin were given daily.

In laboratory animals, research has resulted in findings on other phases of special interest. Experiments have shown that the formation of cataracts occurs in severely deficient animals and that the condition can be arrested merely by adding riboflavin to the diet (Day, Langston, Shukers. *J. Nutrition* 9:637, May, 1935).

More recently, studies on anemia at the University of Wisconsin (Spectar *et al, J. Biol. Chem.* 150:75, Sept., 1943) confirmed earlier evidence of the importance of riboflavin in blood regeneration. This linking of riboflavin with nutritional anemia has broad implications. If the results of the work on dogs can be applied to human beings, this reaction may be explanation for observed low-iron requirements in certain human subjects (Leverton, *J. Nutrition* 21: 617, June, 1941). It has, in times past been suggested that ample intake of calcium reduces the supposed requirement for iron. Perhaps the true explanation of the supposed "sparing action" of calcium may be that along with the

* Because of popular objection to the use of "nicotinic acid" by the general public who confuse the vitamin with the alkaloid "nicotine", the National Research Council recommends the use of the terms "niacin" and "niacin amide" in lay literature. The terms nicotinic acid and nicotinic acid amide are recommended for scientific literature.

dietary calcium, riboflavin was supplied in appreciable quantity. This could readily occur since the richest source of calcium is milk, which is likewise the most important source of riboflavin in the dietary. It has been frequently demonstrated with human subjects, that without at least a pint of milk daily, it is difficult to supply enough riboflavin in the diet.

Clinical practice indicates that most cases of cheilosis clear up within several weeks if 3 mg. doses of riboflavin are given three to five times daily. Some forms which do not respond may not be true cheilosis, but rather a symptom quite similar in appearance which occurs in certain edentulous cases.

Review of the more recent reports on clinical tests of the value of vitamin B-complex factors in correction of lesions of the oral soft tissues, including infections of Vincent's type indicates a possible role of these factors but without clear cut evidence. It would seem that beneficial changes may be expected to occur fairly soon after supplementation in the individual case if gross lack has been a dominant factor. If such changes do not take place within a short period, it still cannot be safely assumed that the vitamin was not the responsible factor. The study of vitamin inter-relationship (reviewed in Chap. 6, *Dietotherapy*, Wohl) indicates that one vitamin influences another. For example, in cases of thiamine deficiency, significant loss in vitamin C is observed and there is pronounced disturbance of riboflavin metabolism. Thus, since it is now generally recognized that thiamine deficiency is widespread in this country, it is quite possible that borderline riboflavin deficiencies may exist as a result not only of inadequate riboflavin intake, but also of poor utilization of that vitamin as a result of thiamine deficiency. (Sure, *J. Nutrition* 27:447, June, 1944). There is a definite probability that our need for one vitamin is influenced by our supply of another.

It all comes back again to the need for generous use of protective foods such as milk, eggs, liver, kidney, vegetables, and fruits, both as to variety and as to quantity in order to supply essential nutrients. Recent studies of the vitamin content of tissues show a definite and often high correlation between vitamin content of different tissues. Those rich in some of the B vitamins tend to be rich in all. Even a casual study of the following food sources of the B vitamins for which human standards have been set as compared to our own personal intakes should stimulate us to greater efforts in our choice of foods.

FOOD SOURCES OF THIAMINE, NICOTINIC ACID, AND RIBOFLAVIN

Thiamine	*Nicotinic Acid*	*Riboflavin*
pork	dry yeast	milk
kidney	liver	liver
liver	poultry, dark meat	eggs
tomato	meat	green leafy vegetables
green vegetables	fish	
whole grain cereal	enriched flour	
enriched flour		

An example of the liberal ingestion necessary to supply ample nutrients is furnished by Sure (p.257, *Dietotherapy*, Wohl) when he states that, for the adult, a diet containing a pint of milk, an egg, a quarter pound of pork, beef or dark poultry meat, a generous helping of turnips or mustard greens (from three-fourths to a cupful) and several slices of cheese will take care of the daily riboflavin requirement. The greens may be replaced by fresh lima beans, spinach, lettuce, and the cheese by canned salmon or generous portions of cooked fresh

water fish. A quarter pound of liver, whether it is pork, beef, lamb or veal liver, will furnish the entire daily requirement.

With the increased use of canned foods, the report of their nutritive value (*Nutrition Reviews*, 3:91, Mar., 1945) is of interest. It was found that canned peas and asparagus are particularly rich in thiamine, a 100 gram portion (about ½ cup) would provide 5 to 10 per cent of the daily allowance. Late season pack is 11 to 14 per cent higher than early. Fruits such as pineapple and orange are only fair sources. The richest canned sources of nicotinic acid were the fish products containing 5 to 10 mg. per 100 grams, while the richest vegetable is peas containing only 1 mg. For riboflavin, fish products were also the best sources, with asparagus, peas, and spinach good.

In general, the solids in the can contained 62 to 72 per cent of the thiamine, and 70 to 80 per cent of the riboflavin, yet, the fluid portions of the can contents hold sufficient vitamins to make their utilization worthwhile.

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(Continued in the October, 1945 issue.)

Studies In Dental Nomenclature: A Series

II. PERIODONTAL NOMENCLATURE

By HARRY LYONS, D.D.S., *Richmond 19, Virginia*

IN THE introductory article of this series, several undesirable language trends which prevail generally in dental nomenclature were discussed. Periodontal nomenclature is handicapped not only by these but also by several difficulties peculiar to this sphere. Confusion in periodontal nomenclature is rife partly because of the use of several roots in the development of terms, two prefixes about which much argument prevails, and the use of certain suffixes without due regard to their proper significance. This is strikingly exemplified in the 1937 report of the Nomenclature Committee of the American Dental Association (1) which lists the following groups of synonyms:

(a) pericementum, peridentium, periodontium, peridental membrane;
(b) paradentium, parodontium;
(c) paradentosis, parodontosis, periodontosis;
(d) periclasia, periodontoclasia, parodontoclasia.

Not only are these synonyms a source of confusion but the prevailing disagreement regarding their definitions adds to the chaos. The obvious need for clarification prompted the American Academy of Periodontology to develop and adopt an official nomenclature (2). The report on periodontal nomenclature from the A.D.A. Nomenclature Committee (3), published in 1943 and which is now widely adopted by teachers of periodontology, should also do much towards the attainment of language unity in this field. The basic facts upon which these two reports were developed merit detailed consideration.

Dental terms are rooted from the Latin word *dens* and the Greek word *odous*. The roots *dent* and *odont* are derived from these words. Periodontology pertains basically to the structures which support and surround the teeth. The two prefixes used to designate these structures are *peri-* and *para-*, both of Greek origin. The Academy's official nomenclature makes use of the prefix *peri-*. This

is also recommended by the A.D.A. Nomenclature Committee.

The use of the prefix *peri-* antedates the use of *para-* in the United States. The terms *periodontia, periodontist* and *periodontology* were formulated long before the prefix *para-* was introduced into the periodontal nomenclature of this country. As used in medicine and anatomy, *peri-* designates tissues which surround, encase or ensheathe. The structures so designated may be those which surround or encase at a distance as well as those immediately contiguous. Examples: *pericardium, periosteum, perivascular.*

The prefix *para-* has a much wider use and a number of varying inferences. It is, therefore, of much less value as a definitive prefix. In anatomy, it designates structures which lie nearby or beside. Examples: *parathyroid, parapineal.* In pathology, it may designate involvements of structures which lie nearby or beside, as in the instances of *paracolitis* and *paramastitis,* and it also used to name certain diseases that resemble other diseases, as in the instances of *paratyphoid* and *paradysentery.* In chemistry, it is used to designate related compounds. Example: formaldehyde and paraformaldehyde. The prefix *para-* may also connote an embryologic relationship, as in the case of *parovarium.* In addition, it has special inferences in botany and zoology.

From the foregoing discussion of the prefixes *peri-* and *para-,* it is obvious that the prefix *peri-* is the more accurately descriptive of the two for use in terms that pertain to the structures which surround, encase, and support the teeth.

Of the two roots, *dens* and *odont,* the choice is one based on the desirability of having terms for use in dentistry etymologically pure rather than hybrid. Since the prefix *peri-* is of Greek origin, it would appear desirable to use the Greek root *odont.* On this basis, the *periodont-* system of nomenclature is recommended as the most definitive of the various systems proposed. At the same time, it affords dentistry an etymologically classical group of terms.

The suffixes which may be employed in the construction of terms for use in this sphere are quite numerous. Of these, three may be discussed comparatively. The suffix *-itis* indicates *inflammation* of the structure to the name of which it is attached. The suffix *-osis* indicates disease in a broad sense, but more specifically *degenerative disease.* The suffix *-clasia* means *destruction, breaking down* or *breaking off.* All three are of Greek origin. A comparison of the definitions of these suffixes should clarify much of the confusion in periodontal nomenclature. If a periodontal disease is *inflammatory* in character it should be designated as *periodontitis;* if *degenerative* in character it should be designated as *periodontosis.* The term *periodontoclasia* merits careful consideration. By analysis of its component parts, it denotes *destruction* of the structures which surround and support the teeth. Obviously, this can result from a number of different pathologic processes. These may be *degenerative* in nature, as in the atrophic disorders commonly seen in these structures. They may also be inflammatory, acute or chronic, purulent or necrotizing, etc. To reserve this term for any *one* periodontal disease is, therefore, without logical foundation. If it is to be retained in our language, it can be used rationally only as a term to indicate a group of diseases having a common end result: the destruction of the structures which surround and support the teeth. Any effort to give this term a more

limited meaning is to ascribe to it something to which it is not etymologically entitled. It is, therefore, a term of limited usefulness and one which should be used only as a label for the entire group of diseases that destroy the *periodontium.*

The term *periodontium* is subject to marked abuse in periodontal nomenclature. It designates collectively all the structures which surround and support the teeth. These are three in number: the periodontal membrane, the alveolar bone, and the gingiva. It should not be used as a synonym for the pericementum or periodontal membrane because this structure does not surround all of the tooth that is normally surrounded by its supporting tissues. It surrounds only that portion covered by cementum. The gingiva also surrounds the tooth and, in young persons, surrounds the tooth in an area covered by enamel. The alveolar bone likewise surrounds and supports the tooth but in area somewhat less extensive than the periodontal membrane. The term *periodontium* should, therefore, be used to indicate all three structures as a group. To use it for any one of the above noted three structures is to arbitrarily and illogically restrict its definition.

Some authors use an erroneous term, *apical periodontitis,* to indicate inflammatory disease of the periapical area, arising as a consequence of pulp death. Such a designation is incorrect because *periodontitis* denotes inflammation of the *periodontium* and the periodontium includes the gingiva. There can be no such entity as an apical periodontitis since inflammation of the periapical area does not basically or generally involve the gingiva. The correct term to use in such instances is *apical pericementitis.*

Many undesirable terms commonly used in the literature of periodontology have been discussed. These should be deleted forthwith by writers and teachers interested in good language; accordingly, the following terms should be discarded:

Rigg's disease
Vincent's infection
Plaut-Vincent's infection
} uninformative eponyms. (*See* introductory article.)

pyorrhea
pyorrhea alveolaris
} terms designating a symptom but incorrectly used to name a disease.

peridental
peridentium
peridentitis
peridentosis
} hybrid terms.

paradental
paradentium
paradentosis
paradentitis
} hybrid terms using prefix *para-*

parodontosis
parodontium
parodontitis
} terms using prefix *par(a)-*

periclasia } a term made up of a prefix and a suffix without a root; uninformative.

(To be continued in the October issue.)

REFERENCES

1. Dental Terms Recommended by the Committee on Nomenclature of the American Dental Association. *Tr. A.D.A.,* 1937, pp. 261-270.
2. Report of Nomenclature Committee, A.A.P., *J. Periodont.* 12:16-18. Jan. 1941.
3. Report of Nomenclature Committee, A.D.A., *J.A.D.A.* 30:1112-1114. July, 1943.

Editorial

Speaking for the Defense

IN A CURRENT periodical of particular interest to the consumer public the editorials are published under the heading, "Off the Editor's Chest." The following editorial could appropriately be published under the same title because it is being written in a defensive attitude provoked by a recent guest editorial in a give-away dental publication.

The guest editor relates how, in his opinion, dentistry has progressed by a series of spurt and lull periods, the stimuli for the former being furnished by individuals, many of whom contributed new methods and techniques of dental practice. He considers that dentistry has been in a state of lull from a time just preceding the war, up to and including the present, and that it would be well for it to take stock of itself.

Among the ideas suggested for this self-examination by dentistry were these, that dentists have been slow to try and to use new materials developed for them by the manufacturers and that perhaps the manufacturers had progressed more during the period in question than the dentists themselves.

Does progress in dentistry depend upon trying out new materials and technical methods, promptly? In normal times, dentists are "the first by whom the new is tried," as witness the number who flock to the exhibits at dental conventions. But this is war, and dentists at home have been working under a pressure they have never known, and often with no assistance or at best with untrained office personnel. During this period of stress, *known* methods and materials must be used for efficiency and quality of service. The dentist's chief obligation now is to serve the urgent dental needs of the public—thousands being without their regular dentists.

Progress in dentistry is measured by other standards as well as by the adoption of new technical procedures and the use of new materials. The recognition of the mouth as a focus of infection; the Study of Dental Education in the United States and Canada by the Carnegie Foundation and the resulting classification and improvement of dental schools; the organization of the International Association of Dental Research; the recognition of dentistry by the American Association for the Advancement of Science; the establishment of dentistry as one of the health professions; the improvement in current dental literature; none of these spurts in dental progress were mentioned in the guest editorial.

As to the present implied lull period of dental progress—never were dentists more conscious of national interests and of their relation to the health needs of the public. Never have the dental schools been so active in providing better educational facilities for students, and graduate and post-graduate instruction for dentists. Dentists are eagerly taking advantage of opportunities for educational improvement. Throughout the country dental societies have made serious attempts to solve the problem of how to extend the benefits of dental service. Many plans are being tried out as possible solutions. All dentists are seriously interested in the Government's extension of social benefits. There are recent valuable contributions to dental literature in the form of books, reports of dental research, such as *Fluorine in Dental Public Health*, and a live interest in the quality of dental journals. All this in spite of curtailment in the number of dental meetings. Instead of a lull period the present will be known as a period of outstanding progress for dentistry. —G.R.S.

Several of the Lecturers and Many of the Members of the A.A.P. Who Attended the Post-graduate Lecture Course at the W. K. Kellogg Foundation Institute, Ann Arbor, Michigan, May, 1945.

Members in attendance were: Drs. Frank L. Adams, Hunter S. Allen, Edward L. Ball, Frank E. Beube, William Branstad, Charles H. Buck, Bernard S. Chaikin, Clarke E. Chamberlain, Edgar D. Coolidge, Ivan R. Cottrell, S. Edith Davis, Edward L. Gambill, F. Raymond Garvey, Clayton H. Gracey, Dorothy G. Hard, Howard A. Hartman, Roger E. Huntington, Raymond E. Johnson, Jules LeGrand, Arthur H. Merritt, Donald Mosher, Balint Orban, Samuel R. Parks, Thomas E. Prosser, Jr., Harold G. Ray, Celia Rich, E. Ronla Romine, Grace Rogers Spalding, Haidee Weeks, I. M. Wiesetier, Charles H. M. Williams, Myer D. Wolfsohn, Willa Yeretsky.

Notes on the 1945 A. A. P. Postgraduate Lecture Course*

By Roger E. Huntington, *Evanston, Illinois*

The fifth postgraduate lecture course of The American Academy of Periodontology sponsored by the W. K. Kellogg Foundation Institute at the University of Michigan was held April 30-May 2. Dr. Balint Orban and his able committee had prepared a program of general interest to the periodontist. The subjects discussed were presented by medical men eminent in their particular fields. In addition to the generally accepted medical facts, they added the results of recent research. The class carried away the satisfaction of feeling better equipped to continue their specialty as physicians of the mouth.

The first and fifth lecturer was the Viennese anatomist, Harry Sicher, M.D., Associate Professor of Anatomy at the Chicago College of Dental Surgery, Loyola University, who in the limited time at his disposal discussed the comprehensive subject, "Biology of bone." He used the following outline: Bone as a tissue and as an organ; histogenesis of bone; growth of bone tissue; resorption of bone tissue; organogenesis of bones; growth of long bones; growth of the skull; growth of the mandible; influence of bone growth on eruption and tooth movement; functional adaptation of bone; influence of pressure and traction on bone.

Dr. Sicher stressed the importance of a clearer differentiation between bone as a tissue and bone as an organ. Most of the bones of the body develop as endochondral and membranous bones; the resulting bone tissue is the same. A bone, for example, the humerus, in the embryo, consists of cartilage only; in the adult, as an organ, it possesses bone, cartilage, connective tissue, marrow, bloodvessels, and nerves. During its growth the long bone lengthens by the interstitial growth of cartilage, which in turn is replaced by bone tissue; in the transverse diameter, however, it grows by the appositional method. Slightly different patterns of growth were also mentioned. In skull sutures, the cranial pattern is produced by a combination of connective tissue and bone growth without the presence of cartilage. Of more particular interest, however, is the growth pattern of the condylar portion of the mandible which growth in length proceeds in the usual interstitial cartilaginous manner of long bone growth, plus its own unique feature brought about by the presence of a fibrocartilage superimposed over the hyaline cartilage and between which cartilage also grows by apposition.

In the interesting discussion of the functional adaptation of bone, Dr. Sicher formulated the reaction of bone to pressure and traction as follows: Within the limits of endurance, pressure or traction will stimulate bone apposition if applied in an area and in a direction which normally resists pressure or traction respectively. As examples, he cited that vertebrae normally subjected to pressure respond with bone formation under increased pressure, while alveolar bone built for traction will resorb even if slight pressure is applied in an area or in a direction not normally subjected to pressure.

Thus, he gave us a vital approach to the subject of bone, quite different from that of the usual static concept of medicine and dentistry.

As a logical sequence to the foregoing, Dr. Joseph P. Weinmann also from Vienna and now Assistant Professor of Oral Pathology at the Chicago College of Dental Surgery, followed with a lecture on the "Pathology of Bone." His original outline included the effect of dietary and vitamin A, B, C, and D, deficiencies, and also the hormonal disturbances known to be produced by the pituitary, thyroid, parathyroid, and sex glands. Dr. Weinmann confined his discussion to those hormones and vitamins which have a direct influence upon bone tissues and bones.

From among the many hormones produced by the hypophysis, the one promoting growth of the skeleton was mentioned. He pointed out that the acromegalic overgrowth of the mandible in hypophyseal giantism and acromegaly results from the presence of the

* The Program for this Course was published in the *J. Periodont.* 16(2):77. Apr. 1945.

fibrous covering of the condyle which acts as a growth center even after general growth has ceased.

By direct action the thyroid furthers skeletal growth while the primary sex hormones inhibit such growth; on the other hand, they both act indirectly by their influence on the hypophysis.

The emergency measure of the parathyroid regulates the calcium level of the blood in mobilizing the calcium from the bone tissues by osteoclastic resorption and also by direct action upon the kidneys effecting the elimination of surplus phosphorus liberated from the skeleton with the calcium. The two distinguishable types of hyperparathyroidism were cited: the one leading to a generalized osteitis fibrosa and the latent form due to renal disturbances resulting in the development of foci of osteitis fibrosa found in areas of localized trauma.

Of the two vitamins mentioned, the following general statement was made, that ascorbic acid influences the formation of the organic matrix of bone, whereas vitamin D is concerned with its calcification.

An evening session rounded out a full day with a timely discussion of "The Metabolism of Calcium in Adolescence" by Dr. J. A. Johnston, Pediatrician-in-chief of the Henry Ford Hospital, Detroit, Michigan. His research covering a period of years included the study of continuous balances throughout the period just preceding and following menstruation in girls which showed that the period of extremely rapid growth preceding the menarche is characterized by a very high retention of calcium, and the deceleration in growth following the menarche by a depression in the ability to retain calcium. The fluctuant nature of the basal metabolism at this time was also brought out, there being a tendency to an elevation in the rate before the menarche and to a depression just afterwards. He emphasized that either of these tendencies might reach pathological levels.

The intake of calcium for this age period is about 1.4 grams but amounts even 20% below this may result in negative balances, according to Dr. Johnston. Vitamin D was shown to be necessary and the amount suggested was higher than that usually given, even up to 1000 units. The calcium requirement would be met by a daily intake of a quart of milk, an egg, and a serving of cottage cheese. If milk were not taken, then there would be an indication for prescribing dicalcium phosphate. Greater amounts than 2 grams are not accompanied by any increases in the retention of calcium.

Glandular effects other than those of the parathyroid were also discussed. As to thyroid activities, either hyperthyroidism or hypothyroidism result in failure to maintain a positive balance, and since both these conditions are more prevalent in the adolescent period than in earlier years, some measure of the thyroid function should be a part of any study of decalcification. Experiments show that large doses of estrogen depress the retention of calcium in the adolescent, but whether this effect might be reversed if the estrogens supplied a defect could not be answered. Clinical estimates are needed to prove this point.

Dr. Johnston stressed the fact that infection is shown to interfere with absorption and retention of calcium, and he stated that this infection need not produce fever. Chronically infected tonsils might be accompanied by a poor retention of calcium, which status could be improved by their removal.

In his summary, the point particularized was that normal calcium metabolism involved considerably more than the provision of an adequate intake; he, therefore, urged a more complete survey of the factors affecting its utilization.

Tuesday morning was chiefly devoted to a comprehensive discussion of the "Disorders of the Blood Associated with Lesions of the Mouth and Throat" by Dr. Frank H. Bethell, Associate Professor of Internal Medicine and Assistant Director of Simpson Memorial Institute, University of Michigan.

The title suggested a study of the abnormal blood pictures accompanying blood diseases, instead however, the greater part of this lecture was devoted to a clinical description of these diseases. Among those given particular attention were glandular fever, acute and secondary agranulocytosis, and the various leukemias.

An outline suggesting the changes in the white cells productive of leukopenia was given as follows: a diminished production due to inhibition, maturation arrest, aplasia of, and infiltration of the bone marrow; increased loss of cells; accelerated destruction; a redistribution by either a retention in the

internal organs or between the tissues and the circulating blood.

Dr. Bethell stated that since persons with disorders affecting the blood and blood forming organs are particularly susceptible to distrubances within the oral cavity, it is a common experience for such patients to seek advice from their dentists. Diseases characterized by the frequent occurrence of early manifestations within the mouth include pernicious anemia, leukemia, agranulocytosis, and the hemorrhagic disorders. Glossitis, occasionally associated with gingivitis, is frequently present in pernicious anemia. It tends to be periodic and is highly refractory to local treatment although it usually responds rapidly to liver therapy. In leukemia, especially the more acute forms of myelogenous and monocytic types, gingival hyperplasia progressing to necrosis and ulceration is a common early complication. Although the disease will ultimately prove fatal, much can be done to improve the patient's comfort by the use of oral therapy together with mild cleaning solutions. Drastic local therapy and especially dental extractions are to be avoided.

Agranulocytosis is an acute disease characterized by a lack of granulocytes in the peripheral blood and infection usually involving mucous membrane surfaces of the orifices of the body. It is the infection which constitutes the most serious hazard in this disease. Gingivitis and pharyngitis which are especially common, are not accompanied by the usual signs of an inflammatory reaction but with an early appearance of a necrotic membrane. Prompt treatment preferably with penicillin is of vital importance in the management of the infection.

The most important hemorrhagic disorders affecting the oral cavity are thrombocytopenic purpura and hemophilia, although bleeding from the gums may occur in a variety of conditions brought about by alterations in the coagulation mechanism or the integrity of the vascular endothelium, an infrequent example being hereditary hemorrhagic telangiectasia. Careful determinations of bleeding, coagulation, and prothrombin times, and a test for abnormal capillary permeability should be included in the study of all patients with gingival bleeding for which no adequate local cause is found. Patients who give a history of spontaneous or excessive bleeding of the gingivae should not be subjected to surgery without an appreciation of the risk involved. If a surgical procedure is imperative, preparations should be made in advance to control hemorrhage. Transfusions of whole blood or plasma have a temporary favorable effect on most hemorrhagic conditions. Local hemostatics such as topical thrombin are of benefit, but only when the agent can be brought into prolonged contact with the site of the bleeding.

Fortunately, this difficult yet highly important subject was presented in another form by the last lecturer of this Course.

In the afternoon Dr. Sicher reappeared, this time to compress into one lecture on "Applied Anatomy" his usual course of six. While his ample illustrations by diagrams and slides demonstrated the importance of anatomical knowledge in its relation to the presence and course of infections, the disturbances of temporo-mandibular articulations, the proper administrations of local anaesthetics, and points of access for emergency operations, all to the head and neck, this discussion for the periodontist proved to be an anticlimax.

A very welcome respite from the foregoing studies came Tuesday evening from the invitation to visit the William L. Clements Library and to listen to a lecture on the subject of "Rare Books" by Dr. Colton Storm, Curator of Manuscripts.

The Library had its origin in the collecting activities of Mr. William L. Clements, who in the 1890's began acquiring rare books, letters, maps, etc., dealing with American history. In 1922 he gave his library to the University of Michigan and in 1923 erected a building in which to house his collection of rare Americana.

Mr. Storm gave as his definition of *rare books*, "those which are important, desirable, and hard to get." During his lecture, many references were made to rare books, such as the Gutenberg Bible which he described as one of the most beautiful books in the world. Major Andre's letter written the day before his execution, now in the Clements library was read by Mr. Storm as were letters from Martha Washington and Paul Revere's wife.

Following the lecture the group was taken into the Rare Book Room where Mr. Storm exhibited some of the rare items in

the collection. These included the "Columbus Letter," a pamphlet of only 8 pages, printed in Rome in 1493; a book printed in 1507 in which is told how America got its name; and perhaps rarest of all, a book giving the first account in English of the earliest English colony in the New World. Equally interesting was John Eliot's translation (1685) of the Bible into the Indian language.

Many rare maps and manuscripts were also exhibited. That part of the Collection relating to the American Revolution is especially notable. The evening spent in the Clements Library is one that will be long remembered by those in attendance.

Wednesday morning was devoted to an exhaustive presentation with lantern slides on the subject of the "Pathology of Arthritis" by Dr. Granville A. Bennett, Professor of Pathology, at the University of Illinois, College of Medicine. His lecture covered such points as the anatomy and physiology of normal joints, the classification of arthritis, the etiologic and pathogenetic considerations, and the gross and microscopic changes in each major type of chronic arthritis.

Dr. Bennett stated that of the many articular disorders which collectively give rise to the present vast amount of crippling disease, two forms of chronic deforming arthritis, rheumatoid arthritis and degenerative joint disease, stand out most prominently. Although the etiology of these two types remains obscure there is general agreement that they are dissimilar from the point of view of genesis and pathology as well as from the clinical signs and symptoms they produce. In his presentation he described and illustrated with lantern slides his own impressions of the manner in which the articular lesions of these two common types of deforming arthritis arise and progress.

Due to the fact that rheumatoid arthritis and degenerative joint disease have little in common except the involvement of the specialized tissues that comprise the articulations, Dr. Bennett referred briefly to certain anatomical and physiological characteristics of these tissues that may influence their susceptibility to injury.

The majority of articulations are simple in structure. They consist of two or more opposing cartilage-covered bone ends that are joined together by a flexible tube of dense connective tissue of the articular capsule. The capsule is lined by an intimal layer of varying microscopic structure that is usually referred to as the synovial membrane. Within the articular space is a small quantity of clear viscid synovial fluid. Some joints, as for example, the knee, are more complex in design due to the incorporation of menisci, stabilizing ligaments, sesamoid bones or other structures having special functions. In close proximity to the joints are similar tissue components such as fascial layers, tendons, tendon sheaths, and bursae. These structures are of the greatest importance in the normal mechanics of the articulations and, in the majority of disease processes, they are either primarily or secondarily affected.

With this brief anatomical outline Dr. Bennett directed attention to certain characteristics of each of the three most important components of the joint, synovial membrane, synovial fluid, and articular cartilage, and noted the manner in which they respond to injury or are affected by the disease processes under consideration. The inner layer of the articular capsule, the synovial membrane, is to be regarded, on both embryologic and histologic evidence, as connective tissue that has undergone certain adaptive modifications in function and structure. An identical tissue lines the bursal cavities and tendon sheaths. Furthermore, morphologically similar tissues are formed as the lining membranes of certain tissue clefts that develop in connective or bony tissues as the result of suitable mechanical stimulation. Thus it is not illogical to regard the articular cavities as enlarged tissue spaces.

The two most important functions that may be ascribed to the synovial lining tissues are these: the regulation of the passage of materials between the blood, lymph, and synovial fluid, and the secretion of synovial mucin. Investigations of these two functions have yielded information supporting the concept that synovial fluid is a dialysate of blood plasma to which mucin is added as the fluid diffuses through the specialized mucin producing cells of the synovial tissues. The small amounts of protein that are present in the synovial fluid can be ascribed to the slight capillary permeability. Such permeability is probably enhanced from time to time as a result of the trauma-

tic injury sustained in ordinary daily use. In contrast to cartilage, the synovial membrane has a marked capacity to proliferate when injured. This characteristic is perhaps most noticeable in the low-grade and long-standing inflammatory changes that characterize rheumatoid arthritis. In this disease the synovial tissues proliferate to cover and replace the cartilage and eventually the joint surfaces are bound together with fibrous or bony tissues.

The cartilage covering the bone ends at the articulation is an elastic, avascular, and cell-poor tissue which in its normal state has a smooth glistening surface. Its chief, if not sole source of nourishment, is the synovial fluid contained within the articular cavity. Compared with other connective tissues, hyaline cartilage has relatively little capacity for regeneration and repair, following injury. Since articular cartilage is the recipient of most of the sudden mechanical stress exerted upon the skeleton it is apparent that the maintenance of an entirely smooth articular surface so essential to normal function, is constantly endangered. It was not only clearly shown that articular cartilage is readily damaged by a host of toxic agents and by acute injuries, but it was equally well demonstrated that it may be damaged by the minor and repeated traumas encountered in everyday activities. In the average person progressive deterioration of the articular surfaces begins almost as soon as maturity develops, occasionally before. Therefore, cartilage, a tissue that is beautifully adapted to meet its functional requirements is at the same time unable to withstand the trauma incident to day-by-day usage. It deteriorates progressively as the age of the individual advances.

In his slides Dr. Bennett reassembled the following observations. A joint is a relatively simple anatomic structure formed of tissues which, in common with bone, are derived from a single embryologic structure. All of these tissues, bone, cartilage, and synovial membrane possess the capacity of forming mucinous material either as an intercellular matrix in the case of bone and cartilage or as an intercellular fluid in the case of synovial tissue. The mucin formed by the synovial tissues accounts for the high viscosity of the articular fluid; a physical characteristic that is responsible for the pronounced cohesive and lubricating qualities of that liquid. In rheumatoid arthritis and other joint lesions of an inflammatory character this constituent of synovial fluid is markedly altered. The synovial intima is loosely textured and highly vascular permitting a free exchange of diffusible substances between the synovial and vascular fluids. Compared with other body cavity membranes, the synovial lining tissues show greater permeability to many substances of widely varied properties such as foreign proteins, dyes, and bacteria. Such enhanced permeability, together with the known vulnerability of synovial tissues to traumatic injury, doubtless contributes to the great frequency of joint involvement in conjunction with many systemic diseases. These characteristics may be of importance also in the genesis of rheumatoid arthritis.

Although the cartilage covering the bone ends is beautifully designed to permit easy motion of the opposing joint surfaces, its scarcity of cells, low respiratory rate, and extremely limited faculty of regeneration, together with the remoteness of any available blood supply, places this highly developed tissue in a biologically disadvantageous position making it vulnerable to a host of noxious agents and to the poorly understood process of tissue aging. The articular lesion of rheumatoid arthritis is a low-grade inflammatory process of unusual persistence. Although it is essentially a proliferative response in synovial tissues it soon leads to cartilage destruction and bone atrophy with an end result that is tragically crippling. By contrast, the articular lesion of degenerative joint disease begins in the articular cartilage. As its name indicates this disorder is brought on by the deterioration of tissue cells and their intercellular substances. The hyaline cartilage is particularly susceptible to the influences of advancing age, daily wear and tear, and perhaps to specific biological imperfections that are as yet unknown.

Many members of this year's class who were fortunate enough to attend the 1942 assembly when Dr. Freiberg of the University of Michigan presented a lecture on "Arthritis," were again impressed with the vigorous attack by medicine upon this vexatious problem.

The last afternoon session opened with the subject of Allergy. Dr. George L. Waldbott, a specialist in allergic diseases discussed

"Allergies in Relation to Dentistry." He explained the mechanism and the pathology of the allergic wheal, the manifestations of allergy, its causes, and the methods available for its diagnosis.

Dr. Waldbott drew a picture of the continuous struggle of the human body against invading foreign substances known as antigens, carried by inhalants, foods, bacteria, and fungi which reach the blood stream by various routes. Usually an excellent mechanism enables the system to protect itself against these foreign antigens by the production of protective antibodies. However, in the allergic individual this mechanism does not function, and instead specifically harmful antibodies, reagins, are produced. These reagins produce a common poison, histamine, which in turn by dilating the capillary blood vessel walls and rendering them more permeable, allows the serum and cellular elements, especially the eosinophiles, to flow into the tissues, resulting in the allergic wheal.

The localization of the wheal establishes the type of disease. The following were enumerated, with many more suggested: urticaria, bronchial asthma, sinus, migraine, eczema, neuritis, gall bladder, spasms in the ureter and in the uterus simulating appendicitis. The causative agents, pollen, epidermals, foods, and fungi are mainly responsible and in the order named. In addition to these are the non-protein materials and such common drugs as aspirin, quinine, and morphine. An allergic condition is usually inherited, but it may also be acquired similarly to the mechanism developed in anaphylactic shock in animals.

References to allergy of the mouth included two types of lesions in the mucous membranes: one due to contact and the other to hematogenous origin. Among the contact agents are amalgam, gold, rosins, chewing-gum, drugs, denture adhesive powders, and nickel, with many others suggested. The most common foods causing the atopic type of lesion are pineapple, citrus fruit, and nuts. Other reactions included severe allergic shock similar to anaphylaxis in animals produced by the injections of novocaine if injected directly into the blood stream; urticaria of the mouth, throat, and tonsils due to drugs, especially aspirin; geographic tongue which may or may not be allergic; canker sores and stomatis, both of which may also be brought on by bacterial allergy.

The final lecture of the course, "The Relationship between Dentistry and Hematology," was presented by Captain George J. Anday, M. C., formerly Assistant Clinical Professor and Director of Laboratory, Loyola University School of Medicine, Chicago.

As a well trained hematologist, Capt. Anday approached his subject with the use of a very complete, colored chart of the reticulo-endothelial system to orientate the class. From this norm he proceeded to the abnormal, and discussed much as Dr. Bethell had done, many of those diseases which are the direct result of blood disorders.

He emphasized the close relationship between dentistry and hematology because of the various oral lesions associated with certain blood diseases, and enumerated the increasingly familiar dental pictures presented in the lesions of severe anemias, pernicious anemia, glandular fever, agranulocytosis, and the acute leukemias. The dentist's opportunity for their early recognition and diagnosis could by prompt medical therapy often save the patient's life.

Explanations of the immediate mechanism productive of the above mentioned diseases are in most cases unsatisfactory and theoretical. Some of the research done includes the possible influence of specific pathology, including that of dental origin. Dr. Anday's studies on the effects of dental pathology upon blood cell formation, led to the discovery that immediately following the extraction of a tooth, be it normal or infected, there is a marked leukopenia developed and this reaction is then followed by a leucocytosis. This reaction is contrary to the one usually following surgical interference and should be taken into consideration if any dental surgery is to be used upon a patient suffering from any of the leucopenic diseases, for a sudden further depression in the white count of such patients might result in death.

Capt. Anday closed his discussion by referring to the many valuable studies made to detect blood changes resulting from specific pathology. Among the writers reporting such studies were the following: Bryan *et al*, Bryant and Polevitzky, Crance, Daland, Epstein, Goldstein, Haden, Hatton, Harvey *et al*, Ivy, Logan, Peterson and Appleton.